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14. ABSTRACT

Objectives: Increased use of explosive devices in recent military conflicts have resulted in, blast overpressure is the primary cause of traumatic brain injury among combat veterans (Owens, 2008). Primary blast injury has been studied extensively in air-containing organs such as the lungs, gastrointestinal tract, and ear due to their increased susceptibility to primary blast (Hooker, 1924), but recent epidemiology shows an increase in the occurrence of head injuries (Martin, 2008). Since there is little information on the intensity of a blast wave needed to cause blast brain

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Report Title

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Materials and Methods: Shock waves were generated that simulated blasts with charge sizes up to 1000 kg of high explosives. The blast exposure to a gyrencephalic animal model (ferret) was isolated to the head by combined abdominal and thoracic protection that reduced blast levels to an order of magnitude below pulmonary injury threshold. The results were scaled to a 70kg human using a biomechanical scaling technique. The outcomes including apnea, meningeal bleeding, and fatalities were analyzed using logistic regressions in terms of applied shock peak pressure and scaled duration.

Results: Increasing severity of blast exposures (either by increasing peak maximum pressure and positive phase duration or both) increases occurrence of injury. Gross necropsy revealed subdural, subarachnoid, and cerebral contusions typically on or around the brainstem though there were no skull fractures for any blast intensity. Risk functions were developed that relate blast intensity to the risk of developing one or more adverse clinical outcomes; mild and moderate/severe meningeal bleeding, initial apnea, and evoked potential signal loss and were scaled for human exposure conditions. For high explosives at close range, the human fatality injury risk for primary blast exposure to the brain was found to be more than twice the pulmonary fatality injury risk. However, the blast level for 50% risk of mild brain bleeding was found to occur at similar overpressure values as the 50% risk of unprotected pulmonary blast injury onset.

Conclusions: This study presents the first injury risk functions for primary blast injuries to the brain over a range of realistic blast exposures. Though the risk assessments show that the blast intensity necessary for 50% risk of fatality from brain injury is much greater than that needed to cause 50% risk of fatality from pulmonary injury in unprotected persons, mild meningeal bleeding may occur at blast intensities below those for threshold pulmonary injury. This injury risk combined with the strong pulmonary protective effects of body armor may explain the recent incidence of mild/moderate TBI in returning service members. The risk functions provided by this study provide the first realistic brain injury risk assessments that can be used to guide clinical assessments of blast injury, design protective equipment, and guide future blast brain injury research.

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BRAIN INJURY RISK FROM PRIMARY BLAST

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Additional Text (removed from abstract)

In recent military conflicts, ballistic protective body armor is very commonly used and has been shown to reduce the pressure seen by the thoracic cavity up to a factor of 50 (Wood, 2010) increasing the pulmonary system's tolerance to blast exposures. Wood et al. hypothesizes this increase in the pulmonary system's tolerance to be the cause of the increased occurrence of brain injury.